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THE

PSYCHOLOGICAL BULLETIN

GENERAL REVIEWS AND SUMMARIES

THE PHYSIOLOGY OF NERVE AND MUSCLE

BY EDWIN B. HOLT

Glenmere, Maine

Two topics are prominent in the papers published in 1918; firstly, the physiology of the "involuntary" or autonomic nervous system, and secondly, the question as to two kinds of innervation (clonic and tonic) of striated and unstriated muscle and as to the nature of tonus. Piéron (36) and von Brücke (7) summarize present views on the latter topic in a comprehensive way, and both papers deserve to be read by anyone desiring a good general idea of the twofold neuro-muscular process. (Piéron's paper, now rather inaccessible, might well be translated.) Higier's monograph (20) on *Vegetative Neurolgoy* is the most comprehensive and readable introduction to the autonomic nervous system that the reviewer has yet seen, containing just about what the psychologist needs to know so far as this knowledge is to be had at present. The monograph by Kempf (25) on *The Autonomic Functions and the Personality* contains little about the physiology of the autonomic system, but is an interesting and important essay in psychology.

Of the still more general papers, Rijnberk's (39) on the "Rôle and Organization of the Nervous System," elementary but up-to-date, comprehensive and capitally arranged, might well be given verbatim in one or two lectures to a class in physiological psychology. The author believes that the heart and the alimentary tract with its glandular appendages possess a considerable degree of sensori-motor autonomy. Adrian (1) gives a clear and compact summary of the work of Keith Lucas and Adrian on nerve-conduction: it is practically a condensation of Lucas's book, *The Conduction of the Nervous Impulse*, with important additional remarks. Adrian

inclines to attribute to the synapse as a region of decrement the functions of inhibition and summation. "The presence of a region of decrement in peripheral nerve is the only condition necessary for the production of peripheral inhibition and summation and these effects depend simply on the normal course of recovery, according to which an impulse set up in the earlier stages of recovery [from a previous impulse] has an intensity less than normal and an impulse set up in the later stages has an intensity greater than normal." The theory of inhibition as due to interference between two series of periodic impulses meeting on a final common path is interestingly discussed (pp. 43–5): and it is worth noting that Adrian and the other investigators of nerve-conduction seldom if ever mention the "inhibitory nerves" and "inhibitory nerve-impulses" which anatomists and neurologists (for example, Gaskell) are apt to postulate. Of course the nerve-impulse as studied by Adrian and Lucas is periodic, an "absolute refractory phase" intervening between any two, unit, "all-or-none" impulses. This presents, doubtless, the "clonic" form of muscular innervation. Whereas the second or "tonic" innervation, while we have as yet almost no direct experimental data on it, is thought by many to result from a non-fluctuating form of nerve-impulse. Of this latter Adrian says: "The evidence is too complicated and, as yet, too inconclusive to be discussed in detail, but it shows that we must face the possibility that there may be some way of producing a continuous activity in certain neurones and certain parts of the muscle, an activity quite unlike the intermittent discharges which form the usual type of nervous impulse." Head's presidential address, "Some Principles of Neurology" (18), is more especially of interest to clinicians than to psychologists. Some diagnostic cautions are urged, with handsome acknowledgements to Hughlings-Jackson, regarding the positive as well as the negative symptoms which may be due to a destructive lesion; as when—"Injury to the pyramidal system was shown, netatively, in the loss of the finer voluntary movements; but the acitivity of the lower centres, released from control, was evident in the spastic rigidity of the paralyzed parts." (One observes, by-the-way, that throughout the literature, the "control" exercised by the cerebral over the cerebellar, and by both over the spinal, neural levels is still referred to in a way that half insinuates a Cartesian homunculus resident in the higher centres. The indisposition to treat this "control" as deriving likewise from afferent or sensory sources certainly evidences limitations in our present understanding

of the nervous system.) In peroration, Head states that three processes "form the main features of the physiological functions of the central nervous system . . . which lead to integration. . . . First of all those impulses potentially of a like sensory quality, are gathered together. Secondly, all impulses capable of exciting sensations of a different quality are rejected by the receptors, which guard each functional level. The third method is manifested in the phenomena of adaptation. . . . No stimulus acting over a long period can remain continuously at the same level of efficiency; it leads to a state increasingly favourable to the appearance of the opposite phase of activity . . . the tendency to biphasic reaction so characteristic of the central nervous system." The passage is pre-Sherringtonian.

The increasing interest of physiologists in the autonomic nervous system is providing new data of which the psychologist will need to take account. The autonomic is not a separate nervous system, since it appears to consist exclusively of motor neurones and it receives its nerve-impulses from the central nervous system (spinal cord or mid-brain). In many cases afferent fibers are located in the same tracts with the efferent autonomic fibers, but these afferent fibers, uninterrupted, enter the spinal cord *via* the posterior roots (where their cell-bodies are found) and their topographical association with the autonomic nerves seems to be merely an anatomical accident: they are simply afferent spinal nerves. The afferent fibers from the viscera all go to the central nervous system, and sometimes do and sometimes do not take their course along (within) autonomic nerve-trunks. "General visceral afferent fibers are found in the IX and X cranial nerves and in the spinal nerves. Their cells of origin are located in the cerebrospinal ganglia. . . . There is no satisfactory evidence that any afferent neurones have their cell-bodies located in the sympathetic ganglia" (Ranson, **37**, pp. 311–2). Nor are all the efferent neurones of the autonomic system unmedullated neurones: the motor neurones from the central nervous system that supply all the innervation of the autonomic, go to that system as medullated fibres, and sometimes travel in the autonomic tracts, uninterrupted and still medullated, to the near vicinity of the organ which they innervate (Higier, **20**, p. 9). It is true, however, that all motor neurones are relayed into non-medullated ("post-ganglionic" or autonomic in the strict sense) neurones at some point before they reach any non-striated muscle. Nor are all the organs which are innervated by the auton-

omic system "visceral," in the familiar sense of that word: the sweat glands, for instance, the erector muscles of the hairs of the skin, and the dilator muscle of the pupil of the eye, are all innervated from the autonomic system. Again, the local sensori-motor functions (such as peristalsis of the intestine *via* Auerbach's plexus, or as, possibly, the heart-beat) are hardly to be classified under either the autonomic or the central nervous system. And, lastly, the distinction between the "two systems" as being respectively voluntary and involuntary can by no means be maintained; as in the case, for one very obvious example, of the sphincter muscle of the bladder.

Such are some of the reasons why the autonomic cannot be accounted a separate nervous system, and why, therefore, it is of interest to such psychologists as care to take into account at all our neuro-muscular organization. Furthermore, it is intimately connected, no doubt, with the emotions. Higier's monograph (**20**) gives an astonishingly compact and yet clear introductory picture of the anatomy, embryology, histology, physiology, pharmacology, and pathology of the autonomic system. Kempf (**25**) devotes one not very long chapter to the autonomic system itself. Ranson (**37**) writes a brief sketch introductory to six further studies by Ranson, P. R. Billingsley, and S. E. Johnson: these are clear and apparently very careful researches into the more minute topography of the autonomic tracts and into the histology of autonomic ganglia, glomeruli, etc. Johnson (**23**) for example, by means of transection and degeneration experiments, shows that the only nerve terminations existing in the autonomic system are of (motor) nerves which have come from the spinal cord or bulb; they terminate, and their impulses are relayed along non-medullated post-ganglionic fibers. There are no commissural autonomic neurones; nor do any sensory neurones running in autonomic nerve-trunks terminate in autonomic ganglia, but all go, uninterrupted, into the central nervous system. Ranson states that "In the anatomical and histological texts we find no hint that the sympathetic nervous system is made up of definite functional groups and chains of neurones as distinct and sharply limited as are any of the conduction systems of the brain and spinal cord. Nevertheless, such is the case; it is even probable that the functional groups and chains of neurones are more sharply limited in the sympathetic than in the central nervous system. The latter is provided with a mechanism for the widest possible diffusion of incoming impulses, while such diffusion does not occur in the former" (**37**, p. 305). These seven papers are all in one issue

of the *Journal of Comparative Neurology*. Orr and Rows (35) conclude, from a rather ingenious study of the degenerative lesions produced by toxic infection of the central nervous system through the lymph as compared with those produced by infection through the blood, that "in the sympathetic ganglionic chain there is no true reflex arc, or in other words that it cannot subserve reflex action without the intervention of the cerebro-spinal axis." And, again, "the consensus of opinion seems to be that the [autonomic] ganglia merely act as reinforcing centers for stimuli from the central axis." More than one-half of Spadolini's contribution (42) is devoted to a critical review of earlier work on autonomic innervation (inhibitory and augmentatory). The author regards sympathetic innervation as preëminently tonic.

It should be mentioned, perhaps, that the motor neurones which emerge from the central to go to the autonomic nervous system form three groups: the cranial (or cranio-bulbar), the thoracico-lumbar (or sympathetic), and the sacral, outflows. Many organs are innervated from two of these divisions—the cranial and thoracico-lumbar, *or* the thoracico-lumbar and sacral. When such is the case the functions of these two systems are invariably antagonistic to each other. Thus the bulbar innervation to the heart is inhibitory, the sympathetic innervation is acceleratory. It is unfortunate that the nomenclature is still not uniform: some authors, as Ranson (37) and Johnson (23), still use the term "sympathetic" as including all three divisions.

The recent developments in regard to the two varieties of neuro-muscular process (the clonic and the tonic) have further tended to obliterate the demarcation between the central and autonomic nervous systems. The classic doctrine taught that the central nervous system carried but one kind of nervous impulse, that this was conducted to striped muscles, and there produced one kind of contraction, the clonic (rapid muscular twitch). Non-striated muscles were innervated by the "sympathetic" nervous system, their contractions seemed to be slow and maintained or tonic, and the "sympathetic" nervous impulse might (or might not) be different in nature from the impulses of the central nervous system. Now it appears that striped muscles exhibit tonic contractions as well as unstriped (although the latter, apparently, do not show clonic contractions), and that the tonic contractions of unstriped muscles are set up by impulses that are sent out from the central nervous system. Beyond the points so far noted, the views regarding the varieties of neuro-muscular process, and of tonus, diverge.

It appears to be now rather widely admitted (after the researches of A. Perroncito, 1902; A. Mosso, 1904; and especially of de Boer, 1913) that there is a tonic (or postural) innervation of even the striped skeletal muscles by unmyelinated fibers from the autonomic system: although the reviewer finds no mention of such an innervation of skeletal muscle in Higier (20) or in Gaskell's *The Involuntary Nervous Systems*. Piéron (36, p. 89), Rijnberk (40), Mansfeld (32), Lukács, and apparently Bottazzi (6) hold this opinion: Dusser de Barenne (2) argues against it, and Cobb (9) from the fact that unilateral division of the abdominal sympathetic chain did not affect the onset of decerebrate rigidity on the operated side, rather disbelieves in the view that the autonomic system affects (postural) tonus. Von Brücke (7) believes that there is some histological evidence for this twofold innervation of skeletal muscle, but that the functional evidence therefor is still inconclusive. It is generally conceded, whether the autonomic system sends fibers to the skeletal muscles or does not, that the motor nerves from the central nervous system send out two kinds of innervation, a clonic and a tonic, to the skeletal muscles (Piéron, 36; v. Brücke, 7; de Boer; Mansfeld, 32; Hunt, 22, pp. 329–30; Betchov, 4, p. 15). Piéron and von Brücke hold that "tonus of rest" is governed by the autonomic, but "tonus of action" by the cerebro-spinal system. Piéron says (p. 88): "The tonic and clonic processes in skeletal muscles are not—energetically—subject to the same laws; do not involve—chemically—a metabolism of the same substances; do not depend—histologically—on the same elements; nor are they—physiologically—set off by the same nervous mechanisms."

Regarding the nature, and the number of kinds of tonus (including contracture), one finds the following views. Piéron (36) holds that the clonic function of striated muscle involves distinct twitches which may occur singly or "fused in a tetanus as typified by the voluntary contraction." It is a property of the myofibril and its carbohydrate metabolism. Tonic shortening of muscle, of which contracture is a pronounced case, is a function of the sarcoplasm of muscle and involves a nitrogen metabolism with production of creatine: it is very economical of energy, and leads to little or no fatigue. The contractured muscle, instead of warming up, grows cool; and a fall of temperature favors tonic contraction. Piéron hints that the tonic nerve-impulse may be "galvanic" rather than "faradic" in type. Von Brücke (7) believes that not only striped but also unstriped muscle manifests both tonus of action and tonus

of rest. He thinks it probable that action tonus involves active innervation (comparable to a mild tetanus) and active muscular metabolism; but deems it undecided whether rest tonus does or does not involve muscular metabolism. He seems to incline to the view that there is a nervously controlled change of length and maintenance thereof without expenditure of energy. As to a *change* of length on such terms the physicists would have something to say. Von Brücke admits the "lock" mechanism of Langelaan to be demonstrated, but the reviewer does not make out whether he accounts it the same as the energyless maintenance of length just mentioned (*cf.* 7, p. 123). Rijnberk, in a very judicious paper (40) stimulates autonomic fibers going to unstriped and (*via* the gray rami) to striped muscles. In some cases he could (tongue, lip) and in others could not (diaphragm, ankle) obtain contraction or even tonic shortening of the muscles. But, he says, these results may not be significant, for if the autonomic fiber subserves only "plastic tonus," "it may well be that its function s exclusively to maintain muscles at a given length, that is to preserve an existing condition, but that it is incapable of bringing about a change." Rijnberk's experiments, though somewhat complicated and involving hypotheses, seem to show that autonomic innervation alone is not sufficient to produce decerebrate rigidity, and he confirms the results of Cobb (9) that the elimination of autonomic innervation has no effect on decerebrate rigidity (p. 737). But he does not conclude with Cobb that therefore the autonomic system does not govern postural tonus; for decerebrate rigidity, he says, is doubtless a spinal tonus. The data are somewhat conflicting, but on the whole Rijnberk coincides with the view of Langelaan that "contractile tonus" is due to impulses from the spinal cord, and "plastic tonus" to impulses from the autonomic system. "Contractile" and "plastic" (tonus), as here used, are probably equivalent to "tonus of action" and "tonus of rest" as used by the authors mentioned above.

Fröhlich and Pick (13) describe a tonic contracture observed in skeletal and the heart muscle, which is induced by poisoning of the peripheral motor nerves at their point of origin, and which seems to involve no metabolism. The toxic application (strophanthin, etc.) has no visible effect, but produces a "*Kontrakturbereitschaft*" of the motor center, which when this center is otherwise stimulated and the corresponding muscles contracted, prevents them from relaxing again. They remain contracted and apparently

at rest (locked). In this state no electrical or metabolic changes can be detected in either the muscles or the nerves involved. Tetanus toxine will produce this contracture-preparedness at the anterior spinal roots, in cold- and warm-blooded animals: strophanthin and calcium will produce it in heart muscle. De Boer and Fröhlich (5) find that contractured heart muscle of the frog (not produced toxically) shows no action-current. But this was under diphasic connection with the string-galvanometer, and they suggest that monophasic connection might give a different result, inasmuch as de Boer has previously found in veratrine contracture of the gastrocnemius of frog, with monophasic conduction, action currents "*von langsamem Verlaufe.*" (Fröhlich and Meyer found no action-current in catatonia induced by suggestion.) Mansfeld (32) contends for a "chemical muscle tonus" maintained by the sympathetic system in addition to the "mechanical muscle tonus" demonstrated by de Boer: the "chemical" tonus, it appears, involves expenditure of physiological energy. This paper is written in reply to a somewhat labored argument by Dusser de Barenne (2) that Mansfeld's experimental evidence for "chemical" tonus is inconclusive (and it is in fact rather indirect evidence). Schmiedeberg (41) finds that an injection of cocaine or novocaine suffices to relax the contracture produced by strophanthus. He believes that the cocaine acts directly on the elasticity of the heart muscle fibers, and that therefore the contracture resides in the muscle fibers themselves and is not (*contra* Fröhlich, Pick, and de Boer) due to any nervous influence. Similarly, tetanus contracture is due to a change in muscle fibers. Schmiedeberg holds that the lock-muscle of Pecten and other molluscs holds the two shells together by its own "elasticity," and that the nervous impulse operates only to relax the muscle. Bottazzi (6) registers his claim to have originated the "duality view of the contraction mechanism of muscle" (myofibril the basis of clonic contraction, sarcoplasm of tonic). He holds that any clonic contraction of striped muscle is followed by a sustained increment of tonus.

Betchov (4) writes on tendon reflexes, tonus and contracture without reference to a possible autonomic innervation. The paper deals mainly with the phenomena of hyperexcitability of reflex centers accompanied by muscular hypotonia, and the reverse. The author argues that hyperexcitability of spinal reflex centers, as found in cases of cerebral and cerebellar lesions, is due not to the absence of any "inhibitory control" normally exercised by the

higher centers, but to the absence of the normal tonic impulses from these centers (hence the hypotonia), in consequence of which the spinal centers, working at lower tension so to speak, are more susceptible to small increments of stimulation (hence the hyperexcitability). The author makes explicit reference to the Weber-Fechner law. Thus hypotonia and hyperreflexia would necessarily be found together; and for the same reason hypertonia and hyporeflexia (as in Parkinson's disease). There occur, however, changes in the elasticity of muscle fibers themselves, and such variations of tonus are of course independent of reflex hyper- or hypoexcitability. The search for "inhibitory" nerve-fibers has been utterly fruitless. Hunt (**22**) disputes Sherrington's principle of the "final common path," contending that the skeletal muscles are innervated by two "physiologically and anatomically" distinct systems of motor nerves, the "paleo-kinetic system" and the "neo-kinetic system." "The paleo-kinetic function represents a more primitive and more diffuse form of movement, which is under the control of the corpus striatum and is subserved by a strio-spino-neural system" (p. 305). This has nothing to do with the autonomic innervation of skeletal muscles previously referred to: although on page 324 the paleo-kinetic innervation is referred to as "sarcoplasmic." The neo-kinetic system is "for the isolated synergic movements of cortical origin." The author finds a "certain harmony" between Head's conception of protopathic and epicritic sensory systems and his own notion of paleo-kinetic and neo-kinetic motor systems. Various clinical illustrations and interpretations follow. To the reviewer this theory seems to be merely fanciful and ambitious. Jordan (**24**) describes a plastic tonus to be found in the body musculature of animals with essentially hollow or tubular bodies (Coelenterates, Holothurians, etc.). The body wall retains for a time any size or shape (within limits) impressed on it (by the body contents, etc.).

An important and interesting paper by Riddoch (**38**) describes the reflex functions of the completely divided spinal cord in man. In this condition, reflex movements which are normally distinct from one another and locally restricted become diffusely combined. A stimulus adequate for some particular reflex sets up a general response compounded of many effects that were originally appropriate each to one particular reflex alone: that is, reflex local signature is abolished. Riddoch calls this a "mass-reflex." There is evidence that in cases of partial translesion the mass-reflex is more apt to be elicited by stimulating the afferents on the more

transsected side. "The influence then exerted on a reflex arc by portions of the nervous system with which it is still connected is one of inhibition or control." The mass-reflex is mainly flexor, a quality which the author inclines to identify with protective or noci-ceptive. "In all cases of spinal injury in which this extensive motor discharge can be evoked, postural activity [which is largely extensor] is abolished and flexion of the lower extremity can be obtained as a protective reflex only. Primary extension reflexes are absent and diphasic movements of flexion and extension of the lower limbs, resembling those of locomotion, are never observed" (p. 354). This last fact is puzzling, since reciprocal innervation of antagonistic muscles is supposed to depend only on the spinal levels immediately involved. Reflex contraction of the external sphincters of the bladder and rectum are the most persistent reflexes of all. The "excessive and violent character" of the protective reflexes becomes "the terror and despair of the sufferer." "It would seem that the increasing degree of depression of postural activity from 'spinal shock' as the animal scale is ascended from dog to monkey and from monkey to man, indicates a progressive pre-spinal [cerebral or cerebellar] dominance in the series, and that it bears a close relation with the assumption of the upright position" (p. 347).

Hoffmann (21) describes the relation of tendon reflexes to voluntary contractions. In order to obtain the "tendon reflex," however, he employs, instead of a mechanical stimulus on the tendon, an electric stimulation of the nerve (electrodes placed on the skin), and records the action-current in the muscle by means of a string-galvanometer. This arrangement gives two distinct action-currents; one that of the direct nerve-muscle stimulation, the other that of a spinal reflex, the electrical stimulus having reached afferent fibers in the nerve-trunk. Yet so far as the mechanical movement of the muscle goes, the two contractions look like only one. This phenomenon is best gotten from the extensor muscles of the foot. If the muscle is at the outset relaxed, the first action-current is stronger than the second (that of the spinal reflex). If the muscle is voluntarily innervated, the second action-current is reinforced, and when the stimuli are feeble becomes stronger than the first. (It seems odd that the voluntary innervation does not reinforce both action-currents alike.) If the antagonist muscle is voluntarily innervated the second action-current (reflex) cannot be elicited. Increased reflex irritability goes with voluntary innervation. With

the patient voluntarily rising on his toes, the action-current of the extensor of the foot can be varied from 40 to 120 fluctuations per second by varying the frequency of faradic stimulation. "Very energetic contraction succeeds in attaining a synchronism between the action-currents and the stimulation when the latter is 150 per second; and this is probably not the upper limit. The nearer one approaches to this the more irregularities and halvings of the rhythm one finds in the resulting curve": showing "a certain inhibition." The author believes that ordinary skeletal muscular contraction and tonus are only quantitatively, but not qualitatively, different. Dreyer and Sherrington (11), working with the isometric myograph, find that reflex contraction is often stronger than the contraction following a similar single break-shock applied to the motor nerve directly. "A single momentary stimulus of moderate intensity, *e.g.*, a break-shock, even though not far above threshold value of stimulation, applied to the afferent nerve of a spinal reflex center, evokes from that center not uncommonly a brief repetitive series of volleys of motor impulses." The frequency of discharge of the reflex center goes to beyond 75 discharges per sec. and "there is nothing to show" that it does not go to 150 per sec. "At frequency-rates up to somewhat above 55 per second, the rhythmic discharge of the reflex center follows the full frequence-rate of the afferent nerve stimulation, the center emitting successive volleys of centrifugal impulses at the same rate as those evoked in and transmitted to it by the afferent nerve." The refractory phase of the spinal reflex (cat) cannot ordinarily be greater than 12σ. Lutz (29), comparing the threshold values of spinal reflex (knee, flexion, and apparently ipsilateral) and nerve-muscle response (foot, extension), finds that the threshold for the former is about twice that for the latter. This, he concludes, supports the view that the synapse is a point of resistance in the conduction path of a reflex arc. In (30) the same author finds that decrease of temperature raises the threshold for both reflex and nerve-muscle response; and increase of temperature does the reverse. The reflex threshold is the more variable with given changes of temperature. At lower temperatures probably fewer neurones respond to the same strength of stimulus, and fewer synapses are able to transmit impulses. Vészi (44) objects, for no visible reason, to Sherrington's "hypothetical mechanism" of resistance and fatigue in the synapse, and argues that ganglion-cells are the points of resistance in reflex paths. He speaks of a refractory state of motor ganglion-cells.

Langley and Hashimoto (27) devote a considerable part of a paper on muscular atrophy to a critical and experimental study of the sources and limits of error in experiments on the atrophy of muscle by the "bilateral denervation method." The "disuse theory" of atrophy has gained wide acceptance, and an alternative theory "that the atrophy is due to the absence of a trophic influence of the nerves, has as yet no definite meaning." The present experiments were made with an eye to the theory recently suggested by Langley, "that the atrophy is due to the continuous fibrillation which occurs in denervated muscle." Fibrillation being the steady play of tremulous contractions in the fibers of a muscle, this theory should seem to imply that the muscle "atrophies" by being worn out. The authors do not pronounce definitively upon this theory, but they conclude from their rather extensive experiments, "that none of the methods of treatment of denervated muscle now in use—passage of galvanic current, production of contraction, passive movements and massage—can have more than a slight effect in delaying muscle atrophy." MacWilliam, in his study of fibrillation in the mammalian heart (31), states that, "Instead of travelling uniformly right through the mass of muscle . . , as under normal conditions, the excitation wave in fibrillation travels most easily along the complexly arranged fasciculi there being an impairment or failure of propagation at most of the inter-fascicular connections. . . . Fascicular dissociation is an essential feature of fibrillation, which is, strictly speaking, a condition of 'fasciculation' rather than 'fibrillation.'. . . The state of fibrillation is rendered persistent by a disturbance in the normal relations of conduction time and refractory period in the cardiac musculature, resulting in the establishment of a mechanism of circulating excitations." Stevens (43) finds, in agreement with Langley's theory, that the wasting of muscle after nerve section "is not due to the cutting off of a supposititious influence, but rather it is due to an incessant fibrillar activity of the muscles, which begins from three to six days after nerve section and persists until the regeneration of the nerve." He disbelieves in either trophic nerve-fibers or trophic nerve-impulses, and favors "the hypothesis that muscular atrophy is due to fatigue caused by fibrillar contractions." The rate of fibrillar rhythm is from 10 to 20 per second. "There seems to be an exact parallelism between the appearance of the contractions and the wasting of the muscle, and between the disappearance of the contractions and the return of voluntary control."

The observations of Berblinger (3) will, if confirmed, modify our present views on the regeneration of peripheral nerves. Immediately after injury, the cells of Schwann are seen actively reconstructing a line of communication between the cut ends of the nerve-fibers: this activity proceeds from both the central and peripheral cut ends of the nerve, but mainly from the former. Any return of nerve function depends upon this line of communication being established before too much scar tissue is laid down. When nerve-trunks are partially but not severely injured, regenerative activity is observed (in young men) all along the peripheral portion of the axis-cylinders. Edinger (12) finds that the nerve-fiber, in regenerating, is assisted in some way by the cells of Schwann, without which it could not regenerate nor, indeed, effect its original growth. Doi (10) finds that regenerating nerve-fibrils do not readily grow except into the sheaths of old degenerated fibers. Marui (33) has a histological study (serial sections) of Mauthner's giant cells as found in the brains of two varieties of fish. He finds that neurofibrils merely pass through the "net" of Golgi; which latter is not a nerve structure but is of glious nature. The neuraxones form an "axone cap" surrounding the Golgi net: this axone cap is a "plexus" of terminal fibers, with ramifications, but not a true net-work. "The contact theory [of neuronic connection] is a histological impossibility." Parts of Marui's description are difficult to follow.

Von Kries (26) has a semi-popular discussion of three theories of muscular contraction; Engelmann's imbibition theory (*Quellungstheorie*), Bernstein's surface-tension theory, and A. V. Hill's two-process conception. The author is eminently friendly towards Hill, and lays his emphasis on Hill's observations. The latter has shown that muscular contraction involves two processes; a non-oxidative contraction process (which yields lactic acid), and an oxidative recovery process. On the second process, like the resetting of a bow-gun, Weizsäcker has built a *Zweimaschinentheorie*. The recovery process, apparently, stores energy for the next contraction, but whether in chemical form, or in a form congruent with the imbibition or the surface-tension theory, is not yet made out. Gunzberg (17) applied faradic stimulation every two seconds to the sciatic nerve of frog, the gastrocnemius muscle being immersed in Ringer's solution. Oxygen bubbled through the perfusing solution, he finds, retards the rise of the "staircase"; CO_2 accelerates it. Now oxygen retards the formation of lactic acid, and CO_2 accelerates it. Therefore the staircase effect may be the result of a metabolic

process necessary for securing a sufficient concentration of this acid. Galletti (14) finds that the least polarizing current which, applied to a nerve, will produce electrotonus at all, will produce both an- and catelectrotonus. Therefore excitation and inhibition are closely related processes. The paper is mainly devoted to a study of the influence of various drugs on electrotonus. Gruber and Fellows (15) find that adrenalin affects muscles undergoing death-changes as it affects fatigued muscles, heightening irritability. Gruber and Kretschmer (16) find that adrenalin counteracts the fatigue induced by the perfusion of fatigue substances (such as sarcolactic acid, lactic acid, and acid potassium phosphate) through the muscle, in identically the same way as it counteracts the fatigue produced normally in active muscles. Langley (28), in a study of the effects of curari, strychnine and other drugs on pre- and post-ganglionic nerve tracts, states that there is "a presumption that curari, strychnine and brucine paralysed the pre-ganglionic nerves by a direct action on the nerve cells." "Recently Burns and Watson . . . speak of guanidin as poisoning synapses like nicotine, and of atropine as paralysing terminal ganglia. The change in nomenclature seems to me unnecessarily confusing. . . . The facts are expressed most simply by saying that nicotine paralyses pre-ganglionic fibers, and atropine paralyses post-ganglionic fibers." This contention against the synapse seems to the reviewer to be unsupported.

Moral (34) ascribes the stronger action of "indifferent" narcotics (alcohol, ether, etc.) at high temperatures, and of the "basic" narcotics (novocaine, salicylamid, etc.) at low temperatures, to the fact that their *Teilungskoefficienten* (ratio of solubility in lipoid to solubility in water) are higher for high and low temperatures respectively. Burge, Neill and Ashman (8), from experiments with narcotics of widely different constitution, advance the theory "that narcosis is due to the direct destruction of catalase by the narcotic, with resulting decrease in oxidation, while recovery from anesthesia is brought about by an increase in catalase due to the increased output from the liver, with resulting increase in oxidation." The Meyer-Overton theory of narcotics as lipoid solvents could in any case apply to the methane series alone. Vészi (45) advances the view that narcotics retard both non-oxidative and oxidative processes in cells by being adsorbed by the lipoids at the surface of the cell and thus lowering the tension between the cell surface and the surrounding medium. This retards the entry of food substances

and the passage out of waste products, and thus slows down the internal processes of the cell. Herzfeld and Klinger (19) believe that there is no free water in living cells, and that diffusion of substances into cells is possible only by the former becoming chemically united to the colloids of the cell. Therefore there is no need of looking for any "physiological permeability" in living tissue.

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SOME RECENT CONTRIBUTIONS TO THE PHYSIOLOGY OF THE AUTONOMIC NERVOUS SYSTEM

BY F. W. CARPENTER

Trinity College

Physiological Changes Associated with Certain Mental States.—Through the work of Cannon and his collaborators we have learned in recent years that emotional excitement brings about, through the autonomic nerves, an increased secretion by the adrenal glands. The substance liberated, epinephrin, is poured into the blood, carried by the circulation to various parts of the body, and here largely through its stimulating effect on the autonomic nervous system prepares the whole organism for vigorous muscular exertion. Among the resulting changes reported by investigators are inhibition of the movements of the alimentary canal, the shifting of blood from the abdominal viscera to the skeletal muscles, increased cardiac vigor, and the mobilization of energy-giving sugar in the circulation.

A somewhat analogous effect of the emotions of fear and rage in respect to the liver has lately been described by Burge and Burge (**6**). Presumably through the stimulation of the autonomic nervous mechanism during the emotional excitement there is formed in the liver an increased amount of catalase, a substance which, when released to the blood stream and carried to the skeletal muscles, appears to augment the oxidation of these muscles, acting possibly as an organic peroxide (comparable in structure to hydrogen

peroxide) liberating atomic oxygen. This physiological reaction is obviously of advantage to the organism in making available more energy for fighting or escaping from danger by flight, actions which, in nature, are often associated with the emotions of rage and fear.

The experiments which led to these conclusions were made on cats confined in a wire cage, and worried for a considerable period by a barking dog. A comparison of the catalase content of the livers of these "fighting" cats with that of the livers of "normal" or control cats (which had not been annoyed by the dog, but which otherwise had lived under the same environmental conditions) showed that the livers of the excited animals contained about ninety per cent. more catalase.

In a subsequent paper Burge (5) gives additional evidence along the same lines. During the early stages of the administration of ether for anesthetizing purposes cats and dogs exhibit marked mental excitement. By prolonging this period of excitement the catalase content of the blood can be appreciably increased. To show that nervous impulses passing to the liver bring about the production of catalase, the splanchnic nerves were stimulated electrically. As a result of the nervous excitation more catalase than before was found in the blood stream.

Stewart and Rogoff (20), as a result of their experiments with cats, call into question the assertion that hyperglycemia, or excess of sugar in the blood, is one of the conspicuous effects of emotional excitement, its production depending upon the secretory stimulation, through the autonomic system, of the adrenal glands. They doubt if so-called emotional hyperglycemia is a constant or even a common occurrence. If it does occur, they believe it is not dependent upon an augmented secretion of epinephrin by the adrenals. In normal cats frightened by a barking dog they were unable to detect any increase in epinephrin, nor could they find any essential difference in the sugar content of the blood after emotional excitement in normal animals as compared with other animals in which the output of epinephrin was prevented by an operation.

Changes in the cerebral circulation were found by Binet (4) to occur in men startled by the discharge of a revolver or the blast of a siren. The results, however, were curiously variable, ranging all the way from congestion to anemia.

Hyde and Scalapino (10) have published a preliminary report on a series of experiments undertaken to ascertain the effect of

different kinds of music upon the heart beat and blood pressure. Phonograph records were employed. In a subject fond of music the minor tones of Tschaikowsky's death symphony caused an increase in cardiac activity and a fall in blood pressure. These effects the experimenters regard as the problable result of "psychic or reflex inhibition of the vagus nerve and vasomotor center." The Toreador's stirring song from Carmen produced a different effect in that the blood pressure was increased, although the pulse was accelerated as before. The change is attributed to reflex action of the accelerator nerve or possibly inhibition of the vagus. A Sousa march, "The National Emblem," gave a still different result. The heart beat became slower with increased blood pressure. A stimulating effect upon the vagus is suggested. The authors refer in their discussion to the probable influence of music, through the autonomic system, also upon digestion, secretion, muscle tone, and respiration, and mention the possibility that the intelligent selection of music may be of benefit in the treatment of certain types of nervous disorders.

The Gastric Hunger Mechanism in Fasting and Fever.—Vigorous peristaltic contractions of the empty stomach are believed to initiate in the walls of that organ the nervous impulses which arouse in the central nervous system the sensation of hunger. These contractions appear to be occasioned by the local motor mechanisms in the gastric wall (myenteric plexus), but are subject to tonic and acceleratory influences through extrinsic parasympathetic fibers (vagus nerve) and inhibitory control by extrinsic sympathetic fibers (splanchnic nerves).

The hunger contractions of the stomach during prolonged fasting were kept under observation by Carlson (7) in a human subject who voluntarily abstained from eating for a period of fifteen days. Fasting men have usually declared that the sensation of hunger disappears after the first three days; but in Carlson's subject the gastric hunger contractions showed practically normal rhythm and intensity during the entire fasting period. The man reported as the "dominant element in consciousness throughout the fast, ideas or thoughts of food and eating," although the hunger contractions were less uncomfortable or painful than those experienced under normal conditions of food taking. The author is of the opinion that fasting men who say they feel no hunger do not recognize as such the sensation produced by the gastric hunger contractions, but regard it as pain or discomfort from gastro-intestinal disorders.

The fasting subject reported, however, a loss of appetite or desire for food, a sensation which Carlson and other physiologists consider quite distinct from hunger. They apply the term appetite to a milder and pleasanter feeling, based in part on memory of agreeable gustatory or olfactory sensations. Appetite can be aroused experimentally by stimulating nerve endings other than those in the muscular coat of the stomach, which are associated with hunger contractions. Such endings are found for example in the lining of the mouth cavity and esophagus. Carlson accordingly attributes the loss of appetite by his subject to the condition of the latter's mouth, which became coated, and developed a disagreeable taste lasting throughout the fast. The unpleasant sensations originating thus are believed to have suppressed the physiological and psychic elements concerned in the pleasurable anticipation of food.

The absence of hunger and appetite in fever is a matter of common clinical experience. Experiments by Meyer and Carlson (14) on dogs show that during fever there is a depression of gastric hunger contractions. From results obtained through sectioning the vagus and splanchnic nerves, the conclusion was reached that this depression was probably due to the lowering of the vagus tone by the fever or the bacterial toxins associated with it; but the authors do not regard this as a complete explanation of the absence of hunger during a prolonged period of fever.

Anatomical and Histological Structure.—The introduction by Ranson (15) to a series of studies on the structure of the autonomic system by himself and his associates includes a good review of the terminology in present use by physiologists and anatomists, and very properly emphasizes the desirability of approaching the morphological study of the system from the functional point of view. The series of five papers by Ranson and Billingsley (2, 3, 16, 17, 18) has to do with the structural details of the cervical sympathetic trunk, the superior cervical ganglion and its branches, the thoracic sympathetic trunk, the rami communicantes, and the splanchnic nerves.

Langley's contention, based on physiological evidence, that all neurone terminations in autonomic ganglia are those of preganglionic fibers having a cerebrospinal origin, is borne out by the histological studies of Johnson (11). After an operation designed to bring about the degeneration of a group of preganglionic fibers, examination of the related sympathetic ganglia in frogs showed a

complete disappearance of the terminal spirals and pericellular networks normally in functional connection with the ganglion cells (cell-bodies of postganglionic neurones). These endings, which are the only types occurring in the frog's autonomic ganglia, appear therefore, to be exclusively those of fibers originating in the spinal cord. There is no evidence that commissural neurones, *i. e.*, intrinsic connector neurones confined to the sympathetic nerves and their ganglia, are present in the frog.

In agreement with recent work of physiologists indicating that the sensory nerves supplying the digestive tube are distributed largely to its muscular coat, nerve endings of sensory type have been found by means of methylene blue intravitam staining in the outer (longitudinal) muscle layer of the stomach and intestine, and in the adjacent serous coat external to it (Carpenter, **8**). These endings were not observed in other regions of the wall of either stomach or intestine. The terminal structures take the form of skeins and nets in the stomach of the cat, and of tuft-like endings in the small intestine of the dog. They are so placed that they would presumably be affected by vigorous peristaltic movements giving rise, in the stomach, to hunger pangs, or by severe distension or contraction of the muscle tunic in either organ producing pain. Their central connections have not been determined, although the sensory fibers in the intestine have been traced to the myenteric plexus.

General.—The monograph by Dr. Heinrich Higier (**9**), of Warsaw, entitled *Vegetative Neurology*, has been translated into English by Dr. W. M. Kraus, and published in this country. The volume deals with the anatomy, physiology, pharmacodynamics, and pathology of "the sympathetic and autonomic nervous systems." The unfortunate synonymity which exists in neurological nomenclature has led to the employment of terms by the author and translator somewhat confusing to the reader accustomed to the terminology now generally accepted by physiologists, that proposed by Langley. Higier follows the usuage of many pharmacologists in applying the name, autonomic, not to the whole system, as is done by Langley, but to the cranial and sacral divisions (the parasympathetic of Langley). His expression for the entire mechanism is "vegetative nervous system." His "sympathetic" division, however, corresponds with that of Langley, since by that term he designates those components which are in functional connection with the thoracico-lumbar region of the spinal cord.

In those chapters devoted to anatomy and physiology the author bases his descriptions and discussions very largely on the work of a group of investigators whose publications have appeared in the German language. The account which he gives of the histology of the system is impaired by the omission of various details (such as those of the ganglionic synpases), the recognition of which is very helpful in understanding the system as a functional mechanism. Indeed, one or two of the figures designed to illustrate the structural relations of the neurones are not only misleading, but histologically incorrect. It is to be regretted that the translator, owing to his absence in the U. S. army medical service abroad, was unable to revise the proofs of the book and make final corrections.

The Autonomic Functions and the Personality by Dr. E. J. Kempf is another monograph appearing in the same series. The theory about which the book centers is thus expressed in the author's words:

"Whenever the autonomic or affective sensori-motor apparatus is disturbed or forced into a state of unrest, either through the necessities of metabolism, or endogenous, or exogenous stimuli, it compels the projicient sensori-motor apparatus to so adjust the receptors in the environment as to acquire stimuli having the capacity to produce adequate postural readjustments in the autonomic apparatus. In this manner, only, the disturbance of function may be neutralized. The constant tendency of the autonomic apparatus is to so organize the projicient apparatus into a means as to acquire a maximum of affective gratification with a minimum expenditure of energy or effort."

While it is beyond the scope of this review and the competency of the present reviewer to discuss either the validity or the value of this theory as a working hypothesis for the psychologist and psychiatrist, a few comments on one or two of the morphological and physiological considerations on which it is based may not be out of place. In support of his views the author lays stress on the Mosso-DeBoer theory that the skeletal muscles are supplied with sympathetic as well as cerebro-spinal fibers, and that it is through the sympathetic innervation that tonus in voluntary muscles is maintained. The anatomical basis for this theory is found in the observations of Botezat (*Zschr. f. wissen. Zool.*, 1906) and Boeke (*Ana. Anz.*, 1909; summary of his work in *Anat. Anz.*, 1913). These authors describe fine, non-medullated fibers ending in small motor end plates on certain striated muscles in various vertebrates.

It is held that the fibers are unconnected with the medullated cerebro-spinal fibers distributed to the muscles, the assumption being that they are autonomic elements. It cannot, however, be said that the view that the skeletal muscles throughout the body receive this double innervation has gained general recognition among neurologists.

On the physiological side the evidence appears to be conflicting as to the direct influence of the autonomic nervous system on the tonus of voluntary muscle. For example, the recent experimental work of de Barenne (1) and Lopez and v. Brücke (13) gave results opposed to such a view, while Riesser (19), on the other hand, obtained pharmacological evidence in its favor.

It may be added that the facts of vertebrate embryology do not, in the reviewer's opinion, bear out the author's conception of the priority in development of the autonomic apparatus over the cerebro-spinal ("projicient") apparatus. Nor can the inclusion of the diaphragm and its innervation in the autonomic mechanism (in support of the James theory of the origin of the emotions) be defended upon morphological grounds, however far the author may be justified in making such a classification for psychological reasons.

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